Anthracnose Disease of Walnut- A Review

Mudasir Hassan, Khurshid Ahmad

Department of Plant Pathology, Sher-e-Kashmir University of Agricultural Sciences and Technology of Kashmir, Shalimar Campus, 191 121, Jammu & Kashmir, India

Abstract— Walnut (Juglans regia) an important commercial dry fruit crop, is attacked by several diseases causing economic damage and amongst them walnut anthracnose caused by Marssonina juglandis (Lib.) Magnus has posed a serious threat to this crop in India and abroad. Walnut anthracnose results in reduction in quantitative parameters such as size, mass and actual crop of nuts, failure in metabolic processes in leaves and change in biochemical indices. Premature loss of leaves results in poorly-filled, low-quality, and darkened kernels. The disease initially appears on leaves as brown to black coloured circular to irregularly circular spots. These spots eventually enlarge and coalesce into large necrotic areas. Later on these infected leaves turn vellow and drop prematurely. Infection of anthracnose disease on leaves occurred at relative humidity above 95 per cent and severity of infection was not influenced by temperature between 10-32°C but was significantly reduced below 10°C. Anthracnose of walnut has been reported to be caused by Marssonina juglandis (Lib.) Magnus, with Gnomonia leptostyla (Fr.) Ces. and de Not as its perfect stage reported that acervuli produced by fungus appeared early in the season as small black specks on the lower surface of diseased leaves. The pathogen(G. leptostyla) reportedly perpetuates primarily on infected leaf debris, and ascospores produced in perithecia act as the primary inoculum during spring. Burying (ploughing in) the fallen leaves in autumn and winter ,pruning of infected twigs and branches and adequate nitrogen fertilization has been recommended for the management of walnut anthracnose as well as under planting walnut saplings with annual and perennial legumes has been shown to increase foliage nitrogen content. Different formulations of mancozeb, dithianon, flusilazole and copper fungicides controlled anthracnose disease.

Keywords— Walnut, Anthracnose, Marssonina juglandis, Perpetuates, Management.

I. INTRODUCTION

Walnut (Juglans regia L.) is economically an important dry fruit crop which belongs to family Juglandaceae. It

originated in Iran from where it was distributed throughout the world (Arora, 1985). It is mainly grown in china, USA and Iran, whereas India stands seventh in production accounting upto 2.14 per cent of the world walnut production (Anonymous, 2010). In India, walnut is grown in Jammu and Kashmir, Arunachal Pradesh, Himachal Pradesh and Uttarakhand. In J&K, Walnut is grown in Badrawah, Poonch, Kupwara, Baramulla, Bandipora, Ganderbal, Budgam, Srinagar, Anantnag and other hilly areas occupying an area of 83,219 ha with an annual production of 20,873 tonnes (Anonymous, 2012). Jammu and Kashmir State has attained a special place in the international trade of walnuts contributing about 98 per cent of the total production in India (Sharma, 2012). Its cultivation plays a significant role in the economic profile of the farmers living in hilly and backward areas, where economic condition of the people is extremely fragile (Anonymous, 2012).

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Walnut fruit is consumed as a dry fruit and is also used for preparation of bakery products, confectionary and oils. Walnut shells are used in glue and plastics as well as in dusting and solution making for cleaning and polishing surfaces (Bal, 2006). Walnut wood and even its leaves are usable in wood and veneer industry, dying, pharmaceutical and food industries (Zamani et al., 2011). Among all nuts, walnut fruit is rich in protein, oils including omega-3 fatty acids, vitamins and minerals with excellent flavor and rich source of energy (Rana et al., 2007). Its alpha linolenic acid has substantial cardio protective effects as it increases the ratio of high-density lipoprotein cholesterol to total cholesterol, reducing inflammation and improving arterial function (Hu et al., 1999; Diousse et al., 2001; Patel, 2005). It contains 'melatonin' an antioxidant produced by pineal gland and responsible for inducting and regulating sleeps (Reiter et al., 2005). It also reduces the incidence of cancer delays neurodegenerative diseases of aging (McGranahan and Leslie, 2012).

Among the major biotic factors, the important fungal diseases include walnut anthracnose (*Marssonina juglandis* (Lib.) Magnus), root and crown rot [*Phytopthora cactorum* (Lebert and Cohn) Schrot], branch wilt [*Hendersonula*

infection on nuts results in premature fruit drop (Worste and Beineke, 2001).

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toruloidea Nattras], ringspot [Ascochyta juglandis Blotshauser.), downy leaf spot [Microstroma juglandis (Berenger) sacc.)], heart rot [Polyporus squamosa Huds. ex Fr.) Fr.], powdery mildew [Phyllactinia guttata (Wallr. ex Fr.) Lev.; Microsphaera extensa (Cke and Peck)] besides stem canker and die back diseases caused by fungi like Cytosperma leucosperma (Pers. ex Fr.) Fr., Nectria galligena (Bres.), Fusarium solani (Mart. Sacc), etc (Sharma and Sharma, 1999; Anonymous, 2013). Amongst them, anthracnose is the wide spread foliar disease of Juglans spp. and the fungus attacks leaves, nuts and shoots of the current season growth (Berry, 1977; Belisario et al., 2008). Symptoms develop on the on leaves and fruits as irregular necrotic areas that are often surrounded by small chlorotic halos. The disease causes premature defoliation, slows down plant growth, reduces quantity and quality of nut crops, thereby resulting in huge economic loss in the walnut cultivation regions of the world (David, 1997; Belisario et al., 2001; Van Sambeek, 2003; Kalkisim, 2012).

Occurrence and economic importance

Walnut anthracnose or black spot/blotch has been reportedly considered as most serious fungal disease of black walnut (J. nigra L.) and Persian or English walnut (J. regia L.) as well as other species of genus Juglans throughout the walnut growing regions including North and South America, Europe, Iran and other Asian countries (Behdad, 1991; Belisario, 2002; Belisario et al., 2008; Salahi et al., 2009). In India, Kaul (1962) reported the occurrence of walnut anthracnose disease for the first time from Kashmir valley. Hassan (1979) reported the occurrence of walnut anthracnose in Iraq while as, Werner (1994) reported from poland. It is indigenous to North America (Todhunter and Beineke, 1984), economically important in the main production areas of Italy and Hungary (Belisario, 1992; Pinteret al., 2001) and most widespread and dangerous disease in Bulgaria (Tsanov and Roshev, 1976; Kalkism, 2012). Saremi and Amiri (2010) reported that this disease caused 60-80% yield losses in quality and quantity in Iran. Walnut anthracnose results in reduction in quantitative parameters such as size, mass and actual crop of nuts, failure in metabolic processes in leaves and change in biochemical indices (Shirnina and Kotljarova, 2000). Premature loss of leaves results in poorly-filled, lowquality, and darkened kernels (Black and Neely, 1978; Zamaniet al., 2011). Walnut anthracnose infection results in reduction in nut yield which varied from cultivar to cultivar (Pinter et al., 2001; kalkism, 2012). Early

Symptamotology

The symptoms of the walnut anthracnose are mainly observed on current year leaves, twigs, fruits and rarely on shoots. The disease initially appears on leaves as brown to black coloured circular to irregularly circular spots. These spots eventually enlarge and coalesce into large necrotic areas. Later on these infected leaves turn yellow and drop prematurely however severe infection and leaf drop usually occurs late in the season (Black and Neely, 1976; Todhunter and Beineke, 1984; Belisario, 2002; Kalkism, 2012). Berry and Frederick (1997) observed symptoms on leaves, fruits and branches which appear as dark brown spots, more or less circular, usually bordered by a vellow ring. In later stage, spots merge to form large dead areas which usually results in leaf defoliation. Saremi and Amiri(2010) observed the characteristics variation in the anthracnose diseased spot during leaf development. Spot shape and area varied from several mm to several cm, from oval to round shape and often surrounded by a yellow halo. They further observed that the infection of leaves was severe in late summer and some infected trees became defoliated. The disease also affected fruits and nut meat since nut from diseased trees showed dark and shriveled meat and necrotic spots.

Zamani et al. (2011) observed that the walnut anthracnose caused by M. juglandismay appear on green outer layer of fruits in the form of circular black or brown stains while as the disease spots on leaves appear as dark brown, more or less circular spots bordered by a yellow ring which vary from 1/16 to 5/16 inch in diameter. These individual spots later on coalesce and form large necrotic areas. Leaf infection usually results in defoliation but sometimes the infected leaflets remain attached to the tree for much of the growing season. This fungus also appears on thinner branches in the form of oval lesions or irregular circles with brown color tending to grey and with reddish brown peripherals. They further reported that at the middle of season, black points appear on the upper part of the infected leaves bearing reproductive organ of fungi. These organs produce bicellular spindle-shaped conidia somewhat tending to limber (embowed) shape. Worste and Beineke (2011) also observed that the symptoms of walnut anthracnose develop on current year leaves, nuts and stem as irregular necrotic areas, which are usually less than 5mm in diameter, and are often surrounded by small chlorotic halos.

Causal pathogen

Anthracnose of walnut has been reported to be caused by Marssonina juglandis (Lib.) Magnus, with Gnomonia leptostyla (Fr.) Ces. and de Not as its perfect stage (Sogonov et al., 2008; Dastjerd and Hassani, 2009; Anonymous, 2013). Sharma and Sharma (1999) reported that acervuli produced by fungus appeared early in the season as small black specks on the lower surface of diseased leaves. Conidiophores were hyaline, short, simple, elliptical, and one celled packed together in a small layer bearing conidia at the tips. The conidia were variously shaped being straight, ovoid, falcate or with only one end rounded and the other pointed. There was one septa, and two cells are unequal with prominent oil globules and measured 15- 26×2 -5 µm. They further reported that brown coloured perithecia developed on fallen leaves which were immersed in the leaf tissues while the beak protrudes considerably on to the leaf surface. These were amphigenous, solitary, scattered, globose, reddish brown with long cylindric beak. The beak measuring 140-170 µm and 25-40 µm in breadth, while the globose base has a diameter of 120-125 µm. The inner cavity of the perithecia was lined with club shaped to fusiode asci while as the asci were hyaline aparaphysate, 8 spored, measuring $56-62 \times 14$ -16 µm. The ascospores were hyaline, fusiode, straight to slightly curved septate and measured 15-19×4-5 um. Saremi and Amiri (2010) isolated M. juglandis on potato dextrose agar (PDA), corn meal agar (CMA) and nutrient agar (NA) fromleaves, fruits and foliage. They also observed that isolated fungus produces minute black fruiting bodies called acervuli in which conidia were colorless, usually crescent-shaped, and divided by a crosswall into two approximately equal cells. Perithecium with 380- 450 µm in length and 150-260µm breadth neck like structure with $19.5-24.5 \times 7.2-8 \mu m$ ascospore size. Salahi et al.(2007) isolated as a streak single spore on oat meal agar while Jamshidi et al.(2012) isolated M. juglandis from the leaf discs bearing acervuli by transferring germinating macroconidia from 2% water agar to 39% potato dextrose

agar added with 7g/litre of oatmeal. Dastjerdi et al. (2009)

isolated G. leptostyla (anamorph: M. juglandis) on oat meal agar and corn meal agar media. Kalkism (2012) isolated G.

leptostyla on potato dextrose agar (PDA) from the walnut

(Juglans regia L.) leaves showing typical symptoms and

identified as G. leptostyla according to optimal growth of

G.leptostyla occurred at 22°C and pH 5.4.

Physiological studies

Fayret and Parguey (1976) reported that production of ascospores of G. leptostyla occoured at 10°C. Perithecia remain immature at 20°C in which the multinucleate prosporophyte grows normally and produces a syncytium from which the ascosporophyte originates, but differentiation of the ascogenic phase is heat inhibited. They further observed that the cold is necessary for the evolution of initial dicaryotic cells, which are the true carpospore cells. These cells and the ascosporophytic phase have special physiologic requirements as regards temperature. Matteoni and Neely (1979) reported that growth of M. juglandis was maximum at 22 °C and at pH 5.4 whileas sporulation was maximum at 26°C with pH 6.8 on oatmeal agar and the optimum temperature for germination of ascospores and conidia were 26°C and 24°C, respectively. Microconidia were not produced at temperature less than 10°C and did not germinate. They further observed that the light reduced vegetative growth, but promoted production of conidia and acervuli. Production of fertile perithecia were observed in vitro after incubating crosses of 2 mating types in darkness at 10°C for up to 3 months. Belisario et al. (2008) while investigating the mean diameter of colonies grown in vitro at 22°C and sporulation of 191 isolates of G. leptostyla grouped by site of collection, were compared observed that the isolates that grew significantly more slowly were from sites with colder early springs and higher altitudes. Acervular conidiomata were abundantly produced by all isolates at 22°C in darkness after 21 days, while productions of protoperithecia were noticed within 2 months by most isolates, under the same conditions. Production of conidiomata was observed at 20 and 25°C while as after 2 months, protoperithecia were present in most isolates at 20°C, very few at 25 and 15°C, and no production was recorded at 10 and 30°C after 2 months. Fertile perithecia with asci and ascospores were produced, after 3 months at 10°C in darkness. They further noticed that the ascocarp diameter, width of asci and length of ascopores of in vitroproduced perithecia were larger than those of perithecia produced in nature. The latter showed a neck length longer than in vitro-produced perithecia. Salahi et al.(2009) reported that oat meal agar to be the best artificial media for growth and asexual reproduction of the M. juglandis. Dastjerdi et al.(2009) noticed that the isolates of G. leptostyla produced acervular type of conidiomata at 21°C temperature, under photo-period of 16-hours light : 8-hours darkness) after 18-21 days of incubation period. Jamshidi (2011) reported that colonies exposed to light produced acervuli faster and in a denser way on optimum pH of 5.8. He further reported that fertile perithecia with asci and

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ascospores were produced in vitro after 75 to 90 days at 4°C in darkness. Slow growths of different Marssonina spp. have also been reported by several workers. Zhao et al. (2010) reported that for rapid mycelial growth and sporulation of Diplocarpon mali, potato and carrot dextrose broth (PCDB) and potato and carrot sucrose broth (PCSB) were most favorable. The optimum temperature for mycelial growth and conidial production was 25 °C. Active mycelial growth occurred at pH 5-7, and pH 5-8 was favorable for sporulation. Galeaet al. (1986) reported that lettuce anthracnose pathogen, M. panattoniana, grows at a wide temperature range at 3-26°C and pH 4-5.2. Colony growth was best on potato dextrose agar (PDA) at 20 °C and pH 5.2. Wolcan (1985) reported that sporulation of Diplocarpon earliana causing leaf scrotch of strawberry was best on malt extract agar with peptone and yeast, at 20

Pathogenicity

 ± 2 °C.

In order to prove the pathogenic nature of *Marssonina* spp. Different workers have adopted different methods. Figueiredo and Hennen (1995) proved the pathogenicity of M. salicicola in the laboratory by artificial inoculation with a conidial suspension brushed on to healthy detached young leaves of Salix babylonica and left in the greenhouse. When maintained in wet conditions, leaf symptoms were evident 12 days after inoculation, and threads of conidia coming out from acervuli were produced 15 days later. Nadroo (2006) proved the pathogenic nature of M. coronaria by spraying the conidial suspension on one year old budded potted plant of red delicious apple cultivar. Cline and Neely (1983) proved the pathogenic nature of the of Gnomonia leptostyla byspraying conidial suspension on mature leaves of Juglans nigra and observed macroscopic lesions at 240 h and acervuli formation after 240 h. Dastjerdi et al. (2009) proved the pathogenicity of M. juglandis by spraying the conidial suspension on mature, fully expanded leaflets. Macroscopic brown spots were observed on leaves 16 days after inoculation while acervuli were observed after 24 days while as Neely (1986) proved the pathogenic nature of G. leptostyla by spraying the conidial suspension on mature leaves of walnut seedlings and noticed the development of lesions on leaf surface after approximately two weeks.

Epidemiological studies

Black and Neely (1976) observed that infection of anthracnose disease on leaves occurred at relative humidity above 95 per cent and severity of infection was not influenced by temperature between 10-32°C but was significantly reduced below 10°C. Rosnev and Naidenov

(1986) also reported that *M. juglandis* requires temperature of 15-30°C, frequent precipitation, and humidity over 65 per cent. Matteoni and Neely (1977) observed that incubation period was directly related to infection frequency. Infection was more severe on older leaves and 10 times more frequent with adaxial inoculation.

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Vonica (1970) reported that conidia reportedly cause secondary infections and intensify the disease during summer. Low temperature, rainfall and high relative humidity at the start of growth promoted infection by the pathogen (Andrievskii and Rikhter, 1976; Jamshidi and Salahi, 2009). Kessler (1984) reported that, after initial lesions arising from infections by ascospores in the month of May, the lesion numbers increased through early summer as a result of secondary leaf infection by conidia. He further observed that disease development was maximum in late July and early August, when most defoliation of previously infected leaflets occurred. Hashemi (2005) noticed that relative humidity of 80 percent for 24 hours and temperature in the range of 10-20°C under windy conditions resulted in more than 80 per cent of ascospores released, whereas high humidity and wind during growing season were necessary for occurrence of secondary infections and dispersal of conidia. Belisario et al. (2001) reported that increase in disease incidence during late August to ending September could be related to the increasing number of leaflets bearing fertile acervuli for secondary infections, leading to progressive senescence of leaves.

Perpetuation

Different Marssonina spp. attacking different crops overwinter by different means either by forming sexual fruiting bodies on overwintering leaves, shoots and fruit debris or as conidia. Vucinic (1977) reported the formation of apothecia of M. brunnea at the end of winter on fallen leaves while Sokolova (1975) observed maturation by late march to early april. Harad et al. (1974) reported the formation of apothecium of M. coronaria on overwintering apple leaves infected with blotch, containing ascospores which served as primary inoculums in spring. Anselmi (1979) reported that M. salicicola infecting Salix babylonicaoverwinters by means of small stromata on the edges of cankers or as conidia in the acervuli on branches. Milicevicet al.(2002) reported that Diplocarpon earliana (anamorph M. fragariae), the causal organism of leaf red spot or leaf scorch of strawberry, overwinters in the form of mycelium and produces two types of fruiting bodies (apothecia and acervuli) in early spring. Primary infections are caused by ascospores and conidiospores from the

acervuli while as secondary infections are caused only by conidiospores.

The pathogen(*G. leptostyla*) reportedly overwinters primarily on infected leaf debris, and ascospores produced in perithecia act as the primary inoculum during spring (Vonica, 1970; Black and Neely, 1978; Dimova and Arnaudov, 2008). Veghelyi and Penzes (1990) observed that the fungus overwintered in infected leaves and occasionally in the epicarp of the fruit. Asci were formed by the end of February and ascospores developed during March. Incubation period took 3-5 weeks. After the appearance of the first symptoms, the development of conidia and secondary infections occurred almost continuously until late autumn.

Jamshidi*et al.* (2009) reported the perithecia from fallen leaves. Perithecia had one beak on leaves and up to four beaks on culture media. Perithecium in homothallic isolates had significantly higher diameter and longer beaks than non-homothallic isolates. Saremi and amiri (2010) reported that the fungus commonly overwintered in fallen walnut leaves, infected during the preceding summer in walnut orchards

Disease management

Cultural as well as chemical management has been suggested by various research workers for the management of walnut anthracnose.

Cultural practices

Burying (ploughing in) the fallen leaves in autumn and winter to a depth of 10-15 cm, pruning of infected twigs and branches and adequate nitrogen fertilization has been recommended for the management of anthracnose(Rosnev and Tsanova, 1980; Neely, 1981; Pscheidt and Ocamb, 2014). Zakhov (1980) recommended rouging and planting healthy material. Shevchenko (1981) advocated that the best control in rugged terrain where the chemical control is difficult is the selection of local immune forms of Juglans regia and production of hybrids with J. nigra, J. cinerea etc. Kessler (1985) found that an over winter cover of autumn olive leaves reduced the number of ascospores, the primary inoculum, discharged from infected fallen walnut leaves. He further observed nitrogen fixed and released from autumn olive increases the walnut foliage nitrogen content and reduces walnut susceptibility to secondary infections initiated by conidia released from the primary infections. Neely (1986) suggested that the incidence or severity of anthracnose can be altered through site modification and increase in foliage nitrogen content. Van Sambeek et al. (2003) reported that under planting

herbaceous legumes in walnut plantations could potentially reduce the severity of anthracnose either in response to increased soil nitrogen or by disrupting ascospore dispersal, while as under planting walnut saplings with annual and perennial legumes has been shown to increase foliage nitrogen content. Saremi and Amiri (2010) recommended that eradication of walnut plant residue, especially fallen leaves is very beneficial in reducing disease. Kalkasim (2012) recommended leaf extracts of *Corcus mas* and *Morus nigra* against *M. juglandis*. Neely (1986) suggested that the incidence or severity of anthracnose can be altered through site modification and increase in foliage nitrogen content.

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Resistance of different cultivars

Black and Neely (1978) while artificially inoculating juglans species with G. leptostyla conidia observed that the Hinds (J. hindsii) and Arizona (J. major) walnuts were more susceptible than black walnut (J. nigra). Little walnut (J. microcarpa), Japanese walnut (J. ailantifolia), butternut (J. cinerea), heartnut (J. ailantifolia var. cordiformis), and assorted hybrids, were less susceptible than black walnut while as the clones of English walnut (J. regia) showed the greatest range in susceptibility. Dimova (2007) reported that Izvor-10 is medium susceptible and the index of infesting could reach up to 52.5 per cent in the leaves and 34.5 per cent in the fruit. Belisaro et al.(2008) observed J. sieboldiana and J. cinerea to be highly resistant and both J. nigra and J. hindsii to be highly susceptible to the disease while as J. regia showed an intermediate response of susceptibility to anthracnose. Salahi and Jamshidi (2009) reported that Z67 and K73 cultivars showed more resistance in comparison with others and Z67 was the most resistant one. The cultivars Ser, Vina, Hartley, Ronde de Montignac, Lara and Franquett had moderate to weak resistance and the cultivars Z63, Z60 and Pedro were all susceptible to the disease. While evaluating 15 walnut cultivars for susceptibility to G. leptostyla infection, Arnaudov and Gandev (2009) recorded that only one cultivar "Chandler" was resistant and five cultivars were slightly susceptible, whereas rest were either susceptible or highly susceptible (cvs. "Sheinovo", "zvor 10" and "Slivenski") or very highly susceptible (cvs. "Alososzentivani" and "Seer") with disease intensity ranging from 4.9 to 62.4 percent.

Chemical management

To manage the disease caused by various *Marssonina spp*. in different host crops, the use of chemicals has been suggested by various workers. Rimfeldt (1979) suggested

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that effective control of *M. salicicola* can be achieved with copper oxychloride at 2500ppm, captafol at 1800ppm, and benomyl at 600 ppm while as Anselmi, (1979) recommended Benomyl, maneb and mancozeb against *M. salicicola* infecting *Salix babylonica*. Sharma (2000) recommended protective sprays with broad spectrum fungicides, mancozeb (0.3%) and carbendazim (0.05%) reduced the disease significantly. Further he observed that benomyl, thiophanate methyl, propineb, chlorothalonil, dithianon and ziram were also effective in controlling the disease.

Milicevic *et al.*(2002) suggested that fungicide Folicur M 50 WP (tebuconazole + tolylfluanid) and Kidan SC (iprodione) were the most effective against the leaf spot disease of strawberries (*M. fragaria*). Devappa*et al.*(2006) suggested that chlorothalanil (0.2%), mancozeb (0.2%), hexaconazole (0.1%), propiconazole (0.1%), carbendazim (0.1%) copper oxychloride (0.3%) were highly effective in controlling the black spot of rose (*Diplocarpon rosae*). Thakur and Nirupma (2010) recommended that Indofil M-45 (0.3%), Antracol (0.3%), Indofil Z-78 (0.3%), Bavistin (0.05%), Kocide (0.3%), Tohfa (0.075%) and Copter (0.3%) against premature leaf fall of apple caused by (*M. coronaria*).

Vonica (1970) reported that (3-6 treatments per year) of zineb, dodine, zinc-metiram, phaltan, PMC, maneb, captan and thiram gave effective control of walnut anthracnose while as Reznikova (1977) observed that 2-6 sprays of combined treatment with Bordeaux and urea gave the best control than treatments with 1% Bordeaux alone. Various workers reported that fungitoxicants like carbendazim, benomyl, DNOC, dodine, cupric oxide, zineb, maneb and chlorothalonil proved to be effective against M. juglandis and significantly reduced the disease (Berry, 1977; Kleiner and Bulatova, 1978; Zamani et al., 2011). Neely (1977) reported that soil application of benomyl, reduced the the incidence and severity of the anthracnose of black walnut for several years. Movsesyan (1978) recommended spraying with 0.5% copper oxychloride and 0.4% zineb during the growing period for control of walnut anthracnose. Rosnev and Tsanova (1980) recommended that Bordeaux mixture 1%, Dithane M-45 0.3%, and Dithane cupromixin 0.6% against walnut anthracnose while as Zakhov (1980) recommended that spraying with Bordeaux, at 2% during winter and 1% before flowering and once after flowering against G. leptostyla. While testing various fungitoxicants on 8-year old walnut trees in Poland, Cimanowski et al.(1991) observed that different formulations of mancozeb, dithianon, flusilazole and copper fungicides controlled

anthracnose and leaf spot (Xanthomonas campestris pv. juglandis). Nakova and Dimova (2003) while investigating the effects of 22 fungicides on the mycelial growth and ascospore germination of G. leptostylain vitro observed that the inhibition of both parameters was greatest with the contact fungicides Kuprozine Super (copper oxychloride), Dithane (mancozeb) and Ronilan (vinclozolin), and with the fungicides Corzate (simoxanile), Rubigan systemic (fenarimol), Topsin M (thiophanate-methyl), (hexaconazole) and Fundasol (benomyl). In field tests conducted in Bulgaria, the efficacy of Dithane, Corzate, Topsin M, Anvil and Fundasol against G. leptostyla in walnut was evaluated. The most effective were Anvil and Fundasol (control of more than 90%), followed by Topsin M (control of more than 80%).Zamani et al.(2011) advocated that application of Bordeaux solution in winter and copper fungitoxicants in early spring could be highly effective for controlling the walnut anthracnose.

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